In the claims:

Please amend the claims as follows (all of the claims under consideration, whether or not amended, are presented below for the convenience of the Examiner):

70. **(Allowed)** The method of claim 73, wherein the step of contacting is performed in vitro.

71. **(Allowed)** The method of claim 73, wherein the step of contacting is performed in vivo.

72. **(Allowed)** The method of claim 73, wherein the B7-2 is human B7-2.

73. **(Allowed)** A method for blocking binding interactions of B7-2 with CD28 or CTLA4 on an immune cell, comprising contacting the immune cell with an antibody that recognizes the polypeptide shown in SEQ ID NO:2 to thereby block the binding interactions of B7-2 with the CD28 or CTLA4 on the immune cell.

74. **(Allowed)** The method of claim 73, wherein the antibody is a polyclonal antibody.

75. **(Allowed)** The method of claim 73, wherein the antibody is a monoclonal antibody.

76. **(Allowed)** The method of claim 73, wherein the antibody is a chimeric antibody.

77. **(Allowed)** The method of claim 73, wherein the antibody is a humanized antibody

78. **(Allowed)** The method of claim 73, wherein the antibody is a human antibody.

79. **(Allowed)** The method of claim 73, wherein the antibody is a $F(ab')_2$ or an Fab' fragment.

80. **(Amended)** The method of claim 73, wherein the antibody is produced by a hybridoma selected from the group consisting of: ATCC accession number HB 11688, ATCC accession number HB 11687, and ATCC accession number HB 11686.

81. **(Allowed)** A method for inhibiting proliferation of a T cell comprising contacting a cell bearing B7-2 an antibody that recognizes the polypeptide shown in SEQ ID NO:2 to thereby inhibit the proliferation of the T cell.

82. **(Allowed)** The method of claim 81, wherein the step of contacting is performed in vitro.

83. **(Allowed)** The method of claim 81, wherein the step of contacting is performed in vivo.

84. **(Allowed)** The method of claim 81, wherein the B7-2 is human B7-2.

85. **(Allowed)** The method of claim 81, wherein the antibody is a polyclonal antibody.

86. **(Allowed)** The method of claim 81, wherein the antibody is a monoclonal antibody.

87. **(Allowed)** The method of claim 81, wherein the antibody is a chimeric antibody.

88. **(Allowed)** The method of claim 81, wherein the antibody is a humanized antibody

89. **(Allowed)** The method of claim 81, wherein the antibody is a human antibody.

90. **(Allowed)** The method of claim 81, wherein the antibody is a $F(ab')_2$ or an Fab' fragment.

91. **(Amended)** The method of claim 81, wherein the antibody is produced by a hybridoma selected from the group consisting of: ATCC accession number HB 11688, ATCC accession number HB 11687, and ATCC accession number HB 11686.

92. **(Allowed)** The method of claim 81, further comprising contacting the cell with an additional immunosuppressive agent.

93. **(Allowed)** A method for inhibiting cytokine production by a T cell comprising contacting a cell bearing B7-2 with an antibody that recognizes the polypeptide shown in SEQ ID NO:2, thereby inhibiting cytokine production by the T cell.

94. **(Allowed)** The method of claim 93, wherein the step of contacting is performed in vitro.

95. **(Allowed)** The method of claim 93, wherein the step of contacting is performed in vivo.

96. **(Allowed)** The method of claim 93, wherein the B7-2 is human B7-2.

97. **(Allowed)** The method of claim 93, wherein the antibody is a polyclonal antibody.

98. **(Allowed)** The method of claim 93, wherein the antibody is a monoclonal antibody.

99. **(Allowed)** The method of claim 93, wherein the antibody is a chimeric antibody.

100. **(Allowed)** The method of claim 93, wherein the antibody is a humanized antibody

101. **(Allowed)** The method of claim 93, wherein the antibody is a human antibody.

102. **(Allowed)** The method of claim 93, wherein the antibody is a $F(ab')_2$ or an Fab' fragment.

103. **(Amended)** The method of claim 93, wherein the antibody is produced by a hybridoma selected from the group consisting of: ATCC accession number HB 11688, ATCC accession number HB 11687, and ATCC accession number HB 11686.

104. **(Allowed)** The method of claim 93, further comprising contacting the cell with an additional immunosuppressive agent.

105. **(Allowed)** A method for downregulating an immune response comprising administering an antibody that recognizes the polypeptide shown in SEQ ID NO:2 to a subject, such that an immune response is downregulated.

106. **(Allowed)** The method of claim 105, wherein the antibody is administered prophylactically.

107. **(Allowed)** The method of claim 105, wherein the antibody is administered therapeutically.

108. **(Allowed)** The method of claim 105, wherein the subject is a human subject.

109. **(Allowed)** The method of claim 105, wherein the antibody is a polyclonal antibody.

110. **(Allowed)** The method of claim 105, wherein the antibody is a monoclonal antibody.

111. **(Allowed)** The method of claim 105, wherein the antibody is a chimeric antibody.

112. **(Allowed)** The method of claim 105, wherein the antibody is a humanized antibody

113. **(Allowed)** The method of claim 105, wherein the antibody is a human antibody.

114. **(Allowed)** The method of claim 105, wherein the antibody is a $F(ab')_2$ or an Fab' fragment.

115. **(Amended)** The method of claim 105, wherein the antibody is produced by a hybridoma selected from the group consisting of: ATCC accession number HB 11688, ATCC accession number HB 11687, and ATCC accession number HB 11686.

116. The method of claim 105, further comprising contacting the cell with an additional immunosuppressive agent.

117. **(Amended)** The method of claim 116, wherein the agent is an anti-B7-1 antibody.

118. **(Amended)** The method of claim 116, wherein the agent is an immunosuppressive drug.

119. A method for blocking binding interactions of B7-2 with CD28 or CTLA4 on an immune cell, comprising contacting the immune cell with an antibody that recognizes the polypeptide shown in SEQ ID NO:23 to thereby block the binding interactions of B7-2 with the ligand on the immune cell.

120. A method for inhibiting proliferation of a T cell comprising contacting the T cell with an antibody that recognizes the polypeptide shown in SEQ ID NO:23 to thereby inhibit the proliferation of the T cell.

121. A method for inhibiting cytokine production by a T cell comprising contacting the T cell with an antibody that recognizes the polypeptide shown in SEQ ID NO:23, thereby inhibiting cytokine production by the T cell.

In the Specification

Please amend the substitute specification filed on August 6, 2001 as follows:

at page 6, please replace the paragraph beginning at line 20 with:

Another embodiment of the invention provides antibodies, preferably monoclonal antibodies, specifically reactive with a peptide of a novel B lymphocyte antigen or fusion protein as described herein. Preferred antibodies are anti-human B7-2 monoclonal antibodies produced by hybridoma cells HF2.3D1, HA5.2B7 and HA3.1F9. These hybridoma cells were deposited on July 19, 1994 with the American Type Culture Collection at 12301 Parklawn Drive, Rockville, MD 20852 as ATCC Accession No.HB 11686 (HF2.3D1), ATCC Accession No.HB 11687 (HA5.2B7), and ATCC Accession No.HB 11688 (HA3.1F9).

at page 9, please replace the paragraph beginning at line 3 with:

--*Figure 8* A and B are the nucleotide and deduced amino acid sequences of the human B lymphocyte antigen B7-2 (hB7-2-clone29).--

at page 9, please replace the paragraph beginning at line 12 with:

--*Figure 11*A and B are graphic representations of the proliferation of CD28+ T cells, as assessed by ^{3}H-thymidine incorporation or IL-2 secretion, to submitogenic stimulation with phorbol myristic acid (PMA) and COS cells transfected with vector alone or vectors directing the expression of either B7-1 or B7-2.

at page 9, please replace the paragraph beginning at line 16 with:

Figure 12 A-G are graphic representations of the inhibition by mAbs and recombinant proteins of the proliferation of CD28+ T cells, as assessed by ^{3}H-thymidine incorporation and IL-2 secretion, to stimulation by PMA and COS cells transfected with vector alone (vector), or with a vector expressing B7-1 (B7-1) or B7-2 (B7-2). Inhibition studies were performed with the addition of either no antibody (no mAb), anti-B7 mAb 133 (133), anti-B7 mAb BB-1 (BB1), anti-B5 mAb (B5), Fab fragment of anti-CD28 (CD28 Fab), CTLA4Ig (CTLA4Ig), or Ig control protein (control Ig) to the PMA stimulated COS cell admixed CD28^{+} T cells.

at page 9, please replace the paragraph beginning at line 24 with:

Figure 13 A-B show the sequence homology between the human B7-2 protein (h B7-2) deduced amino acid sequence (SEQ ID NO: 2) and the amino acid sequence of both the human B7-1 protein (h B7-1) (SEQ ID NO: 28 and 29) and the murine B7-1 protein (m B7) (SEQ ID NO: 30 and 31).

at page 9, please replace the paragraph beginning at line 28 with:

Figure 14 A-D are the nucleotide and deduced amino acid sequence of the murine B7-2 antigen (mB7-2) (SEQ ID NO: 22 and 23).

at page 10, please replace the paragraph beginning at line 3 with:

Figure 17 A-C depict flow cytometric profiles of cells stained with an anti-hB7-2 monoclonal antibody, HA3.1F9. Cells stained with the antibody were CHO cells transfected to express human B7-2 (CHO-hB7.2), NIH 3T3 cells transfected to express human B7-2 (3T3-hB7.2) and control transfected NIH 3T3 cells (3T3-neo). The anti-hB7.2 antibody B70 was used as a positive control.

at page 10, please replace the paragraph beginning at line 8 with:

Figure 18 A-C depict flow cytometric profiles of cells stained with an anti-hB7-2 monoclonal antibody, HA5.2B7. Cells stained with the antibody were CHO cells transfected to express human B7-2 (CHO-hB7.2), NIH 3T3 cells transfected to express human B7-2 (3T3-hB7.2) and control transfected NIH 3T3 cells (3T3-neo). The anti-hB7.2 antibody B70 was used as a positive control.

at page 10, please replace the paragraph beginning at line 13 with:

Figure 19 A-C depict flow cytometric profiles of cells stained with an anti-hB7-2 monoclonal antibody, HF2.3D1. Cells stained with the antibody were CHO cells transfected to express human B7-2 (CHO-hB7.2), NIH 3T3 cells transfected to express human B7-2 (3T3-

hB7.2) and control transfected NIH 3T3 cells (3T3-neo). The anti-hB7.2 antibody B70 was used as a positive control.

at page 34, please replace the paragraph beginning at line 32 with:

Particularly preferred antibodies are anti-human B7-2 monoclonal antibodies produced by hybridomas HA3.1F9, HA5.2B7 and HF2.3D1. The preparation and characterization of these antibodies is described in detail in Example 8. Monoclonal antibody HA3.1F9 was determined to be of the IgG1 isotype; monoclonal antibody HA5.2B7 was determined to be of the IgG2b isotype; and monoclonal anibody HF2.3D1 was determined to be of the IgG2a isotype. Hybidoma cells were deposited with the American Type Culture Collection 12301 Parklawn Drive, Rockville, MD 20852, which meets the requirements of the Budapest Treaty, on July 19, 1994 as ATCC Accession No.HB 11686 (HF2.3D1), ATCC Accession No.HB 11687 (HA5.2B7), and ATCC Accession No.HB 11688 (HA3.1F9).

at page 64, please replace the paragraph beginning at line 16 with:

Human $CD28^+$ T cells were isolated by immunomagnetic bead depletion using mAbs directed against B cells, natural killer cells, and macrophages as previously described (Gimmi, C.D., Freeman, G.J., Gribben, J.G., Gray, G., Nadler, L.M. (1993) *Proc. Natl. Acad. Sci USA* 90, 6586-6590). B7-1, B7-2, and vector transfected COS cells were harvested 72 hours after transfection, incubated with 25μg/ml of mitomycin-C for 1 hour, and then extensively washed. 10^5 $CD28^+$ T cells were incubated with 1 ng/ml of phorbol myristic acetate (PMA) and 2 x 10^4 COS transfectants. Blocking agents (10μ

g/ml) are indicated on the left side of Figure 12 and include: 1) no monoclonal antibody (no blocking agents), 2) mAb 133 (anti-B7-1 mAb), 3) mAb BB1 (anti-B7-1 and anti-B7-3 mAb), 4) mAb B5 (control IgM mAb), 5) anti-CD28 Fab (mAb 9.3), 6) CTLA-Ig, and 7) control Ig. Panels A-G of Figure 12 show proliferation measured by ^{3}H-thymidine (1μ Ci) incorporation for the last 12 hours of a 72 hour incubation [and Figure 12, panel b, shows] IL-2 production as measured by ELISA (Biosource, CA) using supernatants harvested 24 hours after the initiation of culture.

at page 86, please replace the paragraph beginning at line 28 with:

Three hybridomas, HA3.1F9, HA5.2B7 and HF2.3D1, were identified that produced antibodies to human B7.2-Ig. HA3.1F9 was determined to be of the IgG1 isotype, HA5.2B7 was determined to be of the IgG2b isotype and HF2.3D1 as determined to be of the IgG2a isotype. Each of these hybridomas were subcloned two additional times to insure that they were monoclonal. Hybidoma cells were deposited with the American Type Culture Collection 12301 Parklawn Drive, Rockville, MD 20852, which meets the requirements of the Budapest Treaty, on July 19, 1994 as ATCC Accession No.HB 11686 (HF2.3D1), ATCC Accession No.HB 11687 (HA5.2B7), and ATCC Accession No.HB 11688 (HA3.1F9).